

Rescue of Adult Neurogenesis in Down Syndrome Mouse Model

Josh East¹

¹Department of Psychology, IUPUI School of Science

Down Syndrome (DS) is a disease caused by the complete or partial trisomy of human chromosome 21, which can cause abnormal development, structure, and function of neural networks in the brain. These defects can result in various behavioral, cognitive, learning and memory deficits. Adult neurogenesis in the hippocampal formation, a process vital to certain forms of learning and memory, has also been shown to be impaired in mouse models of DS. The reduction in neurogenesis has been associated with overexpression of *Dyrk1A*, a gene involved in cell proliferation. Here, we will use a well-understood mouse model of DS, the Ts65Dn mouse, to study the effects of Epigallocatechin gallate (EGCG), a known inhibitor of the Dyrk1A protein, on adult neurogenesis. If our hypothesis is correct, EGCG will rescue neurogenesis in Ts65Dn mice to the level of controls. This would demonstrate the potential of EGCG as a safe treatment for the learning and memory deficits associated with adult neurogenesis in individuals with DS.

Mentor: Charles Goodlett, Department of Psychology, IUPUI School of Science